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Survey of Porcine Reproductive and Respiratory Syndrome

*Bryan J. Becker, DVM **
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A swine disease of unknown etiology was described in 1987 by veterinarians and researchers in the United States. Since no known pathogens could be implicated, it was believed to be unique. This decision was based on the disease's severity and duration as well as a combination of reproductive and respiratory signs.

Within three years of its discovery, clinical signs of the "mystery swine disease" were reported in all principal areas of swine production in the United States. Months later, a syndrome similar to the U.S. "mysterious disease" was reported from across the Atlantic Ocean — Germany.

In 1991, four years after it was first described, a break was made through this seemingly bleak situation by researchers at the Central Veterinary Institute, Netherlands. They managed to isolate the virus and sequenced the entire genome, calling it "Lelystad virus," after the locality from which it was isolated.

With this progress, an international symposium on the somewhat less mysterious swine disease was held in Saint Paul, Minn. Participants decided to name the disease Porcine Reproductive and Respiratory Syndrome (PRRS), which was adopted by the International Office of Epizootics.¹

Since its naming, several groups have



This is 3-day-old pig, experimentally infected with PRRSV, exhibits lethargy, chemosis, respiratory distress and a rough haircoat.

isolated the PRRS virus (PRRSV) and have used those isolates to reproduce the disease in growing pigs and pregnant sows. Such studies have helped develop diagnostic tests and have helped make the pathogenesis more clearly understood. New vaccines, new introduction methods for gilts into the herd and depopulation of specific segments of a herd (to break the cycle of infection) have made control of PRRS more of a reality.¹

Clinical Signs

Clinical signs and losses in production vary widely among herds with PRRSV infection. Clinical signs range from inapparent (discovered only after serologic testing) to severe (losses of more than 20% of pig production).² This variability may be the result of a prevailing health status, a different virus strain, management factors or a combination of all three.

The clinical signs of PRRS can occur in all types of swine production systems — from "all-in, all-out" to continuous flow systems.

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All animals appear to be susceptible to infection with PRRSV because clinical signs (reproductive failure and respiratory disease) can be observed in porcine of all ages.

Most PRRS clinical reports in the past have focused on acute, severe disease. Now, chronic and subclinical PRRSV infections constitute the majority of cases.¹

Acute Disease: Initial

The initial phase of PRRS begins at any stage of production and lasts one to three weeks with the disease often rapidly spreading to other stages of production. The typical clinical signs include inappetence, lethargy, depression and fever.

The inappetence that is observed is often termed "rolling inappetence" since it is seen in nearly the whole herd with only 20% to 30% of pigs affected at one time. Rolling inappetence lasts 1 day to 7 days in individual animals. Rarely do animals refuse to feed; usually they decrease intake and feed more slowly.¹

Lethargy and depression can be observed in all stages of production with a decreased libido in boars.³

While fever associated with PRRS is variable among herds, usually less than 30% are affected at one time. Rectal temperature in sows rarely exceeds 40°C; growing and finishing pigs can have temperatures between 40°C and 41°C.

Respiratory distress is one clinical sign seen in the initial phase. Polypnea is occasionally observed in adult pigs but is usually more prominent in younger animals. "Thumping" can be seen in weanling pigs and more often in suckling pigs.

Acute Disease: Climax

Premature farrowings, increased numbers of stillborn, mummified or weakborn pigs and increased preweaning mortality are associated with this 8 week to 12 week phase. Farrowing at 107 days to 113 days gestation, which occurs in 5% to 30% of sows during this phase, is described as a late term abor-

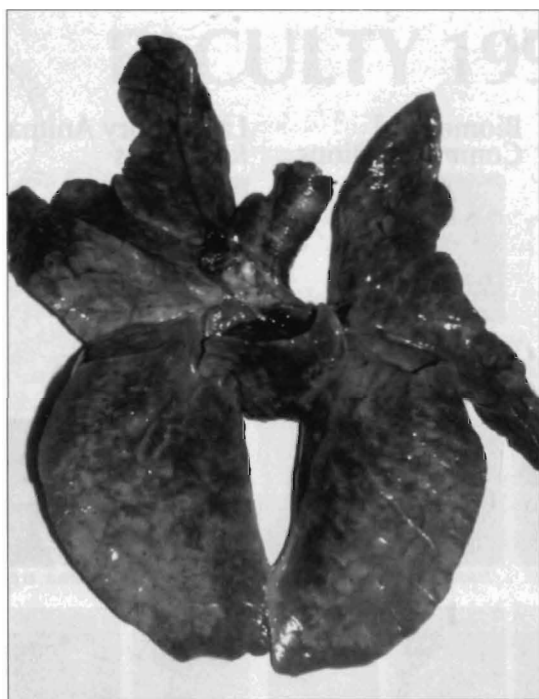
tion.⁴ The farrowings of the breeding herd in this phase contain up to 35% stillborn pigs.^{4,5} Litters may contain 0% to 100% stillborn pigs and fetuses that appear to have died *in utero* less than one week earlier. These large mummies are edematous and can be tan, brown or black. The number of stillbirths peak early in the phase, are gradually replaced by more mummies, then return to elevated numbers of stillbirths late in the phase.⁴ The resulting effect of the increased stillborns and mummies significantly decreases the number of live births per litter during the peak month of the outbreak.¹

Pigs that are born during the climax are often weak, especially if premature. Weakborn pigs often become "starve-outs" or crushed piglets. Piglets also die of respiratory problems and are often seen thumping. Frequently observed are eyelid edema, conjunctivitis and sneezing.⁶

The future for the majority of the piglets in the farrowing house is grim: sickness, fading and ultimately death. Preweaning mortality can average up to 80% on a weekly basis during this phase, which (to remind the reader) lasts 8 weeks to 12 weeks.

Clinical signs in growing and finishing pigs during the climax phase is variable and often complicated with secondary pathogens, including *Hemophilus parasuis*, *Streptococcus suis*, *Salmonella choleraesuis*, *Pasteurella multocida*, *Actinobacillus pleuropneumoniae* and *Mycoplasma hyopneumoniae*. Other complications include more severe primary or secondary viral infections, including swine influenza virus (SIV), pseudorabies virus (PRV), porcine cytomegalovirus (PCMV), porcine respiratory coronavirus (PRCV) and porcine paraymxovirus.⁴

Production flow appears to have an impact on the severity of clinical signs in the climax phase. Producers with "all-in, all-out" sites often experience fewer losses after the initial infection. The prevailing health status of the growing herd is important because most of the growth reduction and mortality that occurs in this phase is due to the secondary infection.



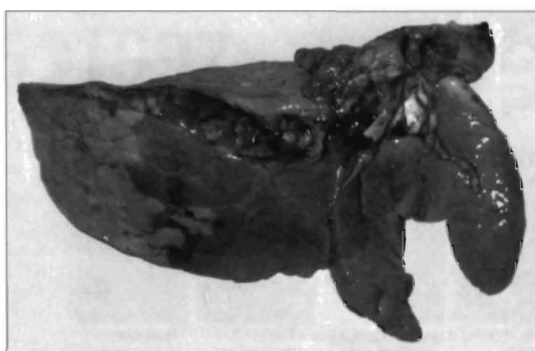
Acute Disease: Final

Reproduction levels almost return to pre-PRRS levels with variable respiratory disease in nursery and grow-finish pigs. This phase may either prelude chronic PRRS or prelude normal, pre-PRRS production levels, depending upon management and production flow.¹

Chronic Disease

The main cause of chronic problems in the sow herd appears to be the recirculation of virus when new seronegative gilts are added to the herd. This explains the importance of gilt isolation and acclimatization, which will be discussed later in greater detail. However, reproduction in most herds returns to normal within 2 months to 6 months of the initial infection.⁵

The chronic effects of PRRS are better described in the nursery and grow-finish areas of production.^{4,5,7} PRRSV has been isolated up to 2.5 years after the initial outbreak.⁷ There has also been grow-finish units with antibody to PRRSV 1 year to 2.5 years indicating chronic infection in these herds.⁸



Top, Lung from a grower pig with severe pneumonia due to combined PRRSV, *Pasteurella multocida* and *Mycoplasma hyopneumoniae* infection.

Left, Lung from a 6-week-old pig experimentally infected with a virulent strain of PRRSV.

The chronic cases of PRRS tend to focus around the secondary infections in the grow-finish units. The result is decreased growth, decreased feed efficiency and increased mortality. The average daily gain (ADG) can be reduced by as much as 15% with chronic PRRS infections.^{4,7} Mortality can often be more than double the pre-PRRS level, at a level of 15% post-weaning mortality, with a cyclical pattern of months between the peaks of the disease.

Subclinical Disease

It is evident with current serology that PRRS has infected more herds than clinical signs have shown. There are probably several reasons for this: virus strain differences, health status of the herd and producers' abilities to recognize and report clinical signs.

Diagnostics

PRRS serology testing follows the same guidelines as serological testing of other swine diseases. First, paired serum samples should be provided to a diagnostic laboratory when evaluating possibilities of a recent infection. Second, the vaccination status and the possibility of passive transfer of antibody

(Continued after the faculty composite on Page 74)

ies should be taken into account when evaluating the results.

An appropriate sample size should be submitted depending upon the expected prevalence of infection, size of the population and the level of confidence that is needed. For example, a sample size of 30 provides a 95% degree of confidence for detecting a prevalence of at least 10%, while a sample size of 10 provides a 95% degree of confidence for detecting a prevalence of at least 30%. In single-site, farrow-to-finish herds, the seroprevalence of PRRS is usually highest in the grow-finish units. Serum from 10 grow-finish animals is usually adequate to determine whether PRRSV has infected the herd. In multi-site production systems, each site is considered a single, separate population requiring individual samples for each.

A negative result on serology can have four interpretations. One, the pig was not infected with PRRSV. Two, the pig was recently infected with PRRSV and has not yet seroconverted. Three, the pig was infected with PRRSV but has become seronegative. Four, the test employed for PRRSV antibody detection was negative because of low test sensitivity or lab error.⁹

Summary of Diagnostic Tests

In the United States and Canada, there are three widely used tests to detect PRRSV antibody in swine serum. These tests are indirect-flourescent antibody (IFA), enzyme-linked immunosorbent assay (ELISA) and serum neutralization (SN).⁹

IFA antibodies (IgG) are present by 7 days to 11 days post infection, peak at 30 days to 50 days post infection, and gradually decrease to undetectable levels by 4 months to 6 months. Post-infection IFA has a high specificity of 99.5% with unknown sensitivity for individual animals (see Table 1). One advantage of the IFA test is that the magnitude of the titer can be measured. The titers of IFA tests are usually reported as fourfold

dilutions starting at 16 to a peak level of 1024. A titer of 16 is considered positive. The IFA tests are subjectively interpreted, so variation among technicians and labs does exist. Due to the high specificity of the test, only strains very closely related to the PRRSV strain used in the test can be detected.

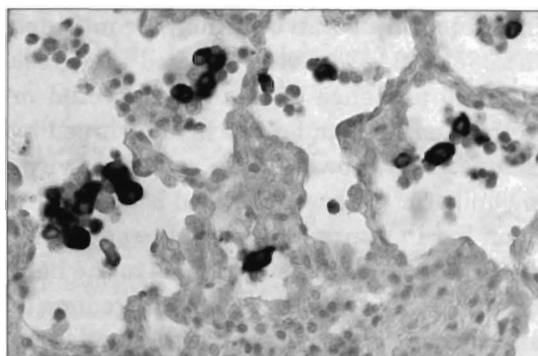
Two different tests, one using the U.S. strain and the other using the Lelystad strain are necessary to detect antigenically diverse PRRSV isolates. Costs of labor and materials are increased by running the IFA for both strains. Another disadvantage is that the test is not automated, so it is time consuming to run on a large scale basis. Therefore, most labs prefer to use the ELISA on the larger volume cases, which they see on a daily basis.⁹

There is another IFA test that is able to detect IgM and more recent infections. This test, however, is not as reliable as the IgG and is problematic with specificity. Therefore, this test is not widely used for diagnostic purposes.⁹

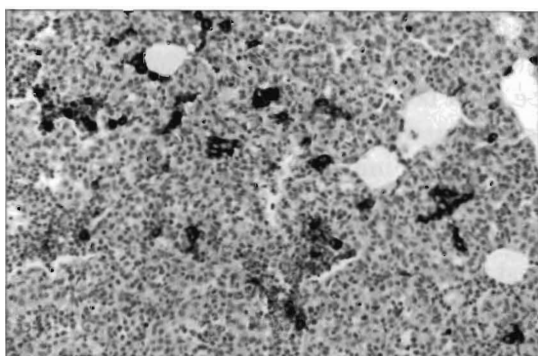
The PRRS ELISA test is reported to have both high sensitivity and specificity, 100% and 99.5%, respectively. ELISA antibodies first appear 9 days to 13 days post infection, peak at 30 days to 50 days and then decline (see Table 1). It is estimated that ELISA antibodies exist at detectable levels for 10 months to 47 months. PRRS antibody formation and decay are similar for both IFA and ELISA tests.

Several advantages of the PRRS ELISA test are: it is automated, a machine reads the results rapidly, it detects both strains of virus (U.S. and Lelystad) and it is licensed by the U.S. Department of Agriculture and AgCanada.⁹

The presence of PRRSV antibodies is determined by measuring the sample to positive ratio (S:P ratio) which is then corrected for nonspecificity. A ratio of 0.4 or greater is considered a positive reading. The S:P ratio might be correlated to the IFA titer, but the manufacturer of the test kits does not currently recommend interpreting the S:P ratio in this manner.⁹



Lung of a pig experimentally infected with PRRSV. PRRSV nucleic acid can be detected with pulmonary alveolar macrophages (dark cells) by *in situ* hybridization.



Lung of a pig from a field case of PRRSV complicated by bacterial infection. PRRSV antigen can be demonstrated within alveolar macrophages (dark cells) by immunohistochemistry.

The PRRS serum neutralization (SN) test is less sensitive than either the IFA or the ELISA. The length of time between the infection and the appearance of antibodies is much longer: 9 days to 28 days. SN titers rise slowly for 2 months to 3 months, with the maximum ranging from 64 days to 256 days, then gradually decline. SN titers are certainly more persistent and have been estimated to be at detectable levels for at least one year. However, the PRRS SN test is not widely used at this time.⁹

Detection of PRRS Lesions by Histopathology

The most characteristic lesion in grow-finish pigs is interstitial pneumonia. The pulmonary lesions noted are: alveolar walls thickened by macrophages and lymphocytes; type II pneumocyte hypertrophy and hyperplasia; and increased necrotic debris and mixed inflammatory cells in alveolar spaces. Some other lesions observed due to PRRSV infection include lymphohistiocytic myocarditis, rhinitis and encephalitis. Formalin-fixed tissue samples of lung, heart, brain, tonsil and nasal turbinates should be submitted for histologic examination on this age of pig.⁹

A recent finding in experimentally transplacentally-infected fetuses, which may be visible grossly, is an umbilical lesion. The umbilicus may be hemorrhagic and edema-

tous with lesions ranging from moderate to severe, segmental to circumferential, characterized histologically by necrosuppurative and lymphohistiocytic arteritis with marked transmural and periarterial hemorrhage. Endothelial lining is often swollen or missing and subendothelial lymphohistiocytic aggregates are common.¹⁰

There are three tests for detecting the presence of PRRSV or antigen within the animal: Direct Fluorescent Antibody (DFA) testing, immunohistochemistry and virus isolation. Another test, Polymerase Chain Reaction (PCR), is most commonly used to detect the presence of virus in semen.³

The DFA is run on fresh tissue (preferably lung or tonsil), which is stained with an PRRSV monolonal antibody fluorescein conjugate. The test is quick and specific, but if the tissue is autolyzed, it may not be very sensitive.

The immunohistochemistry test is more sensitive, but is more expensive and takes longer to run than the DFA, which is complete in 24 hours. Preferred tissues for the immunohistochemistry test are lung and tonsil.

The preferred specimen for virus isolation is serum. The virus can circulate in the blood for 1 week to 6 weeks or longer because the virus is very stable in serum. The virus is quickly degraded in tissues, especially in autolyzed tissue. This explains why the virus is seldom found in aborted fetal tissue.

Serum from animals vaccinated with the modified live virus (MLV) vaccine should not be used because a viremia is produced due to the vaccine. Routine diagnostic tests find the viremia indistinguishable from the wild strains of virus.⁹

The Polymerase Chain Reaction (PCR) test is a very sensitive test. Another point to remember in collecting samples is that the virus is often intermittently shed in the semen. This requires a collection of at least 2 or 3 samples approximately 1 week apart to identify infected, intermittently shedding boars.³

Managing a Positive Herd

When out in the field and faced with a clean up situation, there are many factors to take into account including size of the operation, type of operation, the management level, ability to accomplish “all-in, all-out” flow, multi-site or single-site pig production, method of introducing new breeding animals and serological profile. This article will not attempt to cover all of these factors; however, several recent articles state the key to starting a clean up. The key is to get a serological profile. This profile will identify herd specific pattern of viral transmission and identify areas where virus transmission continues to occur in the herd.^{11,12}

One method for obtaining the serological profile is to first take samples from the different stages of production. These stages include gestating sows, newly weaned pigs (3 weeks to 4 weeks old), grower pigs (8 weeks old piglets) and late finishing pigs (5 months to 6 months old). Ten samples of each age group should be taken, which would yield a 95% chance of finding a 30% prevalence in each stage.

Next, evaluate where the seroconversion is taking place. The breeding herd often continues to circulate the virus in a small number of animals. Without control here, no program will be effective because sows will continue to spread virus among themselves and to their piglets. Three potential causes of continued shedding in the sow herd are

introduction of naive animals, continued introduction of actively infected seedstock and the presence of recently infected or uninfected subpopulations in large breeding herds (>1,000 sows).¹¹ The control of virus shedding in the breeding herd is dependent upon herd closure. It is recommended that no introductions of new animals to the herd be done for a 4 month period. Introduce at one time all the replacement animals you will need for 4 months and close the herd. After the proper isolation scheme is established, the herd can be reopened to new seedstock.

Isolation and acclimatization is best accomplished if off-site facilities are available. If the off-site facilities are not available, it is extremely important to have separate air space, such as no common hallways. The time frame needs to be extended with PRRS to 60 days due to its extended periods of viremia, which can last for 30 days to 45 days or longer. A final directive is that the facilities need to be managed on an “all-in, all-out” basis, with cleaning and disinfecting between groups.¹¹

Other measures that can be taken to reduce losses due to secondary bacterial infection in PRRS herds have been described.¹³ This procedure has been called the McREBEL™ approach, which stands for Management Changes to Reduce Exposure to Bacteria to Eliminate Losses from PRRS. This method, outlined below, is designed to reduce secondary infections, which are often the major problem in the nursery and grow-finish units. The major points of the protocol are as follows: cross foster only during the first 24 hours of life and do not move sows or piglets between rooms. Eliminate the use of nurse sows. Humanely destroy piglets that become sick and are unlikely to recover, also minimize handling of piglets, especially administration of routine antibiotics or extra iron injections. Do not transfer undersized pigs back to rooms containing younger litters and immediately stop all feedback of porcine tissue. Move nursery pigs according to strict “all-in, all-out” principles, allowing 2 days to 3 days between groups for clean-

Table 1. – Serological Tests for Porcine Reproductive and Respiratory Syndrome Virus

Serological Test	Antibody First Detected	Peak Antibody Titer	Decline Antibody Titer	Antibody Undetectable At	Positive Titer	Sensitivity	Specificity
Indirect Fluorescent Antibody (IFA) (Detects IgG)	7days to 11 days PI ^a	30 days to 50 days PI	Rapid	4 months to 6 months PI	≥ 1:16 or ≥ 1:20 ^b	Unknown	High
Indirect Fluorescent Antibody (IFA) (Detects IgM)	5 days PI in 3-week-old pigs; 7days PI in sows	14 days PI	Very Rapid	28 days PI in 3-week-old pigs; 21 days PI in sows	≥ 1:16 or ≥ 1:20 ^b	Unknown	Unknown
ELISA	9 days to 13 days PI	30 days to 50 days PI	Rapid	4 months to ≥ 10 months PI	S:P ratio ≥ 0.4	High ^c	High
Serum Neutralization (SN)	9 days to 28 days PI	60 days to 90 days PI	Gradual	≥ 1 year PI	≥ 1:4	Low	High
^a PI = post infection ^b Depends on the initial dilution used in the IFA test ^c Sensitivity (100%, 35/35 samples) and specificity (99.5%, 413/415 samples); personal communication, Michael L. Snyder, IDEXX Laboratories, Inc.							

ing and disinfecting.¹¹

Two strategies aimed directly at eliminating virus shedding in the nursery and grow-finish units are nursery depopulation and a vaccination program. First, nursery depopulation is implemented when the breeding herd is serologically under control and there is still virus circulating in the nursery. All nurseries are emptied at once and an offsite facility is attained until the animals are marketed. They do not return to the production facility. When the nurseries are empty, they should be washed and disinfected three times using 90°C to 94°C water and a rotation of chemical disinfectants used, such as formaldehyde and phenol-based products. Rooms should remain empty for 2 days to 3 days and the pits should be emptied between wash and disinfect cycles.¹¹

The second strategy of vaccinating nursery pigs eliminates the number of pigs that enter the nursery and then become shedders throughout the nursery and grower phase.

One drawback of the MLV vaccine is questionable levels of cross protection achieved between different PRRSV strains. Another drawback is the difficulty in determining if antibody differences are due to vaccine or to virus.¹¹

Conclusion

There are as many methods available and ways of implementing a PRRS-controlling protocol as there are reports on the syndrome itself. However, the basic knowledge of a particular farm's active virus circulation and an understanding of serological results are both necessary before any clean up process can begin.^{11,12}

One must remember PRRS is a very elusive viral disease. Its effects in a swine herd are not yet fully understood. There remains much to be learned about PRRS. It is the duty of the practicing veterinarian to keep abreast of new developments and to educate

swine producers on the best methods of controlling the disease and reducing its effect on the swine industry. **V**

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Veterinary Students Express Concerns About Economic Future

"Managing Your Economic Future in Veterinary Medicine," the April 1996 financial symposium sponsored by the American Veterinary Medical Association met the expectations of those who attended. Another symposium dealing with this subject would be beneficial according to the participants.

Three major categories, veterinary medical education, career concerns and professional perspectives were divided into 12 subsets of activities and addressed by individual speakers. Each attendee attended breakout sessions to select the 5 issues of highest priority and suggest solutions.

Participants ranked the sessions dealing with "education costs," "companion animal medicine," "debt management" and "curriculum dynamics" as the most valuable.

Student reactions ranged from expressing frustration that "veterinarians think that we are taking loans for more money than we

really need and having a great, fancy life" to "there are many misconceptions in and between current students and practitioners as to the scope of the problem. The economic future is a complex problem as viewed by different segments of the profession with their own priorities and values."

"The problem is large and complex, however, there is a willingness to resolve and correct the issue," said one attendee.

"There is a great dichotomy between veterinary students' perspective and that of the profession. In my discussions with students, they came for immediate solutions while veterinarians philosophized. There is an excellent 'window of opportunity' to bring the students aboard," said one participant.

The proceedings for the economic symposium are published in the July 15, 1996 issue of the *Journal of the American Veterinary Medical Association*. **V**